Boffetta et al. (1) in their multicenter study on exposure to environmental tobacco smoke (ETS) and the risk of lung cancer showed that the odds ratios for spousal and workplace ETS exposures were 1.16 and 1.17, respectively, with no clear dose–response relationships. Although they allowed for intake of fruits and vegetables, they did not allow for intake of saturated and unsaturated fats.

A recent meta-analysis of 51 studies showed that smokers have a statistically significantly ($P \leq 0.0001$) higher intake of saturated fat than nonsmokers (2). ETS-exposed nonsmokers have a statistically significantly ($P \leq 0.001$) higher intake of fat compared with unexposed nonsmokers (3). Many studies have shown an increased risk of lung cancer with increasing consumption of fat, for example Wynder et al. (4). Alavanja et al. (5), in a case–control study of 429 nonsmoking women with lung cancer (comparable in size to the study of Boffetta et al.), showed that the relative risk (RR) for all histologic types of lung cancer comparing high quintiles with low quintiles of saturated fat intake was 6.14 (95% CI = 2.63–14.4; $P$ for trend = .0001).

In the study by Boffetta et al., 78% of the case patients were female and 51% of the tumor histologic types were adenocarcinoma. In light of the evidence given by Alavanja et al., how can Boffetta et al. attribute this very small increased risk to ETS exposure?

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REFERENCES


NOTE

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RESPONSE

Dr. Denson questions our results and conclusions regarding lung cancer risk from environmental tobacco smoke because the estimated risks were not adjusted for intake of fat, in particular, saturated fat. Regarding possible confounding, he quotes the results of a study on women from Missouri by Alavanja et al. (1). However, the authors of that article later corrected their estimates of the risk of lung cancer from intake of saturated fat among nonsmokers (2). The corrected relative risks in the highest quintile of saturated fat intake were either 1.78 (95% confidence interval [CI] = 1.04–3.04, nutrient residual method) or 2.38 (95% CI = 1.35–4.17, nutrient density method), depending on the method of analysis used. Unfortunately, we did not collect information on saturated fat intake and could not con-
trol for the potential confounding effect exerted by this factor. The magnitude of the risk found in the study of Missouri women, however, makes it unlikely that uncontrolled confounding from saturated fat intake explains our results regarding exposure to environmental tobacco smoke.

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NOTES

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